I4 - *Bacterium citri*

United States Department of Agriculture
Bureau of Entomology and Plant Quarantine

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(No. 1 is an introductory and explanatory number.)
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Name of disease: Citrus canker, cancrosis.

Name of pathogen: *Bacterium citri* (Hasse) Doidge
Syns. *Pseudomonas citri* Hasse
Bacterium citri (Hasse) Jehle
Phytomonas citri (Hasse) Bergey et al

Hosts: *Citrus* spp. and numerous relatives. The citrus nursery stock quarantine (No. 19) covers all plants belonging to the Sub-family or Tribe Citratae. According to Dr. W. T. Swingle *B. citri* may attack any plant of the sub-tribe Citrinae, judging by tests up to 1938. *Swinglea glutinosa* cankers abundantly in the Philippine Islands. Plants of several genera in the family Rutaceae and not in the sub-family Citratae have been inoculated with positive results. (4, 5, 13, 17)

Part attacked: Leaves, twigs, larger branches, and fruit. Roots have been inoculated successfully, but with difficulty. (8)

Place of origin: Asia. Found on herbarium material of *Citrus medica* collected in India (? 1827 - 1831) and on *C. aurantifolia* collected in Java in 1842-1844. (6)

Country of first report: Organism described from U. S. in 1915 but disease had been noted in Japan as far back as 1899. (10, 21)

First report from U. S.: Florida, Nov. 1912. Supposed to have been introduced about 1911. (see (3))

Present distribution: Borneo, Ceylon, China, Dutch East Indies, Formosa, Hawaii, India, Indo-China, Japan, Java, Malay States, Mauritius, Mozambique (Portugese East Africa), New Guinea, New Zealand, Northern Territory (Australia) Philippine Islands, Seychelles, Siam. The disease was present in South Africa for a time but was eradicated. Latest reports of the occurrence of citrus canker in the United States are Alabama (1927), Florida (1927), Georgia (1917), Louisiana (1939), Mississippi (1922), South Carolina (1916), and Texas (1938). The disease is not present in the commercial citrus regions of Louisiana and Texas and only a very few scattering infections have been found in these states in recent years, hence eradication should be established as completed before long. The disease may have been eradicated from Northern Territory (Australia). (3, 5, 7, 11, 18)

Methods of spread: Air currents, beating rain, man, animals, insects, etc. Drops of rain or dew on affected organs contain large numbers of bacteria and the disease can be transferred readily by physical or animal agencies from plant to plant.

Nursery stock, fruit or other plant parts serve to carry the disease to new areas. The present distribution of the disease shows its introduction into islands and other areas near ports of call on trade routes radiating from its original home, which may be taken to indicate spread with the fruit. (4, 7)

Losses incurred: In Florida it was said to render infected grapefruit trees worthless inside of two or three months. Owing to its destructiveness and rapid spread, it was necessary to eradicate the disease so quickly that it had no time to demonstrate its power for sustained destructiveness under Florida conditions and on less susceptible hosts under such conditions. (1)

Comparative losses: Where susceptible varieties are grown as in the United States and the Union of South Africa it was necessary to eliminate the disease or it would eliminate profitable production with the citrus varieties being grown. Elsewhere, as in Ceylon where the varieties grown are resistant and conditions less favorable for the spread of the disease, it is controlled by spraying, sanitation, and cultural practices and losses are not severe. (7)

Control Methods: The cheapest control in some areas is accomplished by eradication and strict quarantines. The most satisfactory method of eradication is destruction of diseased trees and burning of the soil surface with a blowtorch. In Ceylon trees are sprayed every week in the rainy part of the growing season and every two weeks the rest of the growing season, diseased parts are removed and burned, and irrigation is handled to stimulate the main growth when infection is less likely to occur. These methods give reasonable control in Ceylon. (1, 2, 5, 7, 20, 21, 22)

Quarantine action: Quarantine No. 19, promulgated Dec. 10, 1914, effective Jan. 1, 1915, prohibits the importation into the United States of all citrus nursery stock, including buds, scions, and seeds, from any part of the world. Effective July 1, 1932, citrus seeds were transferred from this quarantine to the less stringent restrictions of quarantine No. 37. The citrus fruit quarantine, No. 28 issued June 27, 1917, effective August 1, 1917, prohibited the importation of citrus fruits except oranges of the mandarin class (satsuma oranges and tangerines) from a large part of the orient and the Union of South Africa. The citrus fruits permitted entry under quarantine No. 28 are subject to the restrictions of the Fruit and Vegetable Quarantine No. 56 also and hence may be prohibited entry under its provisions. (20)

Description: On leaves the disease becomes apparent as minute yellow-brown, oily, translucent, raised spots, usually visible on the lower surface first. As the spots grow and thicken the lower surface becomes spongy and the upper surface also becomes raised and either smooth or ruptured and spongy. If growth is rapid the spongy growth appears white, if growth is slow the surface is tan or brown. When the spot is ruptured the margin is likely to become watery, glazed, greasy appearing and often somewhat raised and yellowish brown or green in color. The spot is surrounded by a halo of chlorotic leaf tissue with a more or less irregular margin which shades off quickly to the normal colored tissue. On fruit the lesions are much the same as on leaves but usually without the halo and in old stages with the margin likely to be raised more sharply giving a more marked crater like aspect.

After the lesions get old, on any part of the host, they become corky looking and the tissue is hard. Margins are likely to be irregular in whole or in part. The maximum size of the lesions varies with the host and the part affected, mostly ranging from an eighth to a third of an inch in diameter.

Other diseases, such as scab and leprosis, having raised lesions do not have the glazed margin with an oily or greasy appearance surrounding the eruption. While canker lesions do not all have a crateriform appearance, there will usually be some present showing this character and helping to distinguish this disease. Melanose lesions may be raised but remain small and smooth. However, it is difficult for specialists familiar with the disease to determine it on intercepted fruit when the lesions are few and not typically distinctive.

The damage done by citrus canker depends on host susceptibility, weather and state of growth of the host. Grapefruit, sweet oranges, limes, and some varieties of lemon are very susceptible. Varieties of *Citrus nobilis*, including tangerines, king, mandarin and satsuma oranges, are resistant to canker. Three cultivated species of kumquat are highly resistant while a wild species is very susceptible.

Infected fruit may drop. If it matures the lesions disfigure it. The disease may cause more or less defoliation. Lesions may kill young growth. Cankers on older wood may do relatively little direct damage but serve as sources of inoculum during periods when spread is most likely to occur.

The organism will withstand considerable periods of drying. Abundant moisture is the most important factor in the spread of the disease, infection taking place readily under a temperature range between 20 and 30° C.

Abundance of moisture produces rapid growth which is most susceptible and also helps to produce an abundance of bacteria, helps to spread them and results in formation of the film of moisture on the plant surface which is important in enabling the organism to gain entrance without the assistance of wounds.

The organism may persist for years in the soil according to some workers but others believe it is killed out under normal field conditions in a few months to a year or two at the most. The length of time it persists seems to depend on the relative antagonism of other organisms in the soil as well as on the character of the soil and temperature and moisture conditions. (5, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23)

Bibliography:

(1) Berger, E. W., Stevens, H. E., and Stirling, Frank
1914 Citrus canker II
History of Citrus canker, by E. W. Berger
Studies of Citrus canker, by H. E. Stevens
Eradication of Citrus canker, by Frank Sterling
Fla. Exp. Sta. Bu. No. 124, pp. 25-53, Oct. 1914

Brief account of occurrence in the United States of citrus canker. Stevens' paper covers symptoms and uncompleted studies including inoculations and effort to find the pathogen, a fungus being mistakenly thought to be responsible for canker. Spread found to be more rapid during rainy weather. Any part of the tree seems to be susceptible at any age. Eradication recommended. Stirling's paper discusses failure of drastic pruning out and spraying treatment and development of the forced flame burning of trees and soil in order to eradicate the disease. "As I have said before, this disease is by far the worst which has ever yet affected the citrus industry. The leaves, twigs, and fruit become covered with a cankerous growth. The fruit itself seems to be especially susceptible to the disease, and drops soon after becoming diseased. Canker is so deadly that when the tree first becomes infected, in this territory, it is worthless inside of two or three months."

(2) Bertus, L. S.
1931 Spraying against canker of Citrus. Trop. Agriculturist 76: 337-9. 1931

In Ceylon citrus canker may be controlled by weekly spraying with 1% solution of concentrated lime-sulphur or fortnightly applications of a 2% solution, supplemented by removal and burning of all affected leaves and twigs. It is recommended that irrigation should be so managed as to get maximum growth during the drier periods when infection of the susceptible young leaves is less likely to occur.

(3) Doidge, Ethel M.
1916 The origin and cause of Citrus canker in South Africa. U. of So. Afr. Dept. of Agric. Sci. Bul. No. 8, 20 pp. 1916.

Citrus trees imported from Florida during the 1905-6 season and planted in a government experimental orchard showed scab-like spots on the fruit in 1908. The following summer was wet and the disease spread with incredible rapidity. Nursery stock was burned, other trees severely pruned and sprayed five times. In 1910 pruning and spraying were practiced again. The 1911 crop was free from disease but in 1916 the disease reappeared. In 1915 it was shown that *Pseudomonas citri* Hasse was the cause of the disease.

(NOTE: While it is stated that citrus canker was introduced into South Africa from Florida in 1905, this does not accord with the history of the disease in the United States. It seems more likely that the disease was introduced into South Africa from the Orient on some resistant host on which it escaped notice and spread to the stock from Florida. However, this discrepancy has never been cleared up apparently.)

(4) Doidge, E. M.
1929 Further Citrus Canker Studies. U. of So. Africa Dept. Agr. Bu. 51, 31 pp. 1929.

Plants of the sub-tribe Citrineae are the most susceptible usually but a number of genera of other sub-tribes were successfully inoculated. Species of *Toddalia* and *Xanthoxylon* are usually immune or highly resistant. Fagara (*Xanthoxylon*) *capensis* and *Clausena inaequalis* were moderately susceptible in inoculation tests.

The decline of *Bacterium citri* in the soil was found to be much less rapid than indicated by Lee and Fulton. Experiments in the field showed that the organism remained alive in the soil and that trees planted in such soil two years or so later developed canker the first year favorable for canker. It is dangerous to replant infected sites even 4 to 6 years after diseased trees are destroyed.

Canker was carried 6 miles in flood water and infected lemon trees.

Costs of canker inspection are given and the work discussed.

(5) Elliott, Charlotte
1930 Manual of Bacterial Plant Pathogens. 349 pp. June, 1930.

Bacterium citri (Hasse) Doidge, description, synonymy, symptoms, hosts, geographical distribution, and control are given on pp. 112-13, literature is listed pp. 114-121

(6) Fawcett, H. S. and Jenkins, Anna E.
1933 Records of Citrus canker from herbarium specimens of the genus Citrus in England and in the United States. Phytopath. 23: 820-844. 1933.

The earliest collections on which citrus canker was found were on *Citrus medica* from northwestern India believed to have been collected 1827-1831 and on *C. aurantifolia* collected in Java in 1842-1844.

(7) Fawcett, H. S.
1936 Citrus Diseases and Their Control (1st ed. in 1926, canker covered pp. 212-222) 656 pp., 187 figs., some in color. 1936.

Pp. 237-249 Citrus canker is discussed, history, distribution, hosts, symptoms, organism, environment, and prevention. Symptoms and how to distinguish canker from other diseases are discussed in some detail.

(8) Fulton, H. R.
1920 Decline of Pseudomonas citri in the soil, Journn. Agr. Res. 19: 207-223. June 1, 1920.

The decline of P. citri in soil kept under conditions approximating those in the field was so rapid as to suggest that agricultural soils probably can not long retain a dangerous possibility of disseminating the citrus-canker organism. The rapid decline of P. citri is thought to be due to deterious effects of other organisms in the soil, in part at least.

(9) Fulton, H. R. and Bowman, John J.
1929 Infection of Fruit of Citrus by Pseudomonas citri. Journ. Ag. Res. 39: 403-426. Sept. 15, 1929.

Inoculations on young fruits are much more likely to be successful if wounds are fresh when inoculum is applied and if oil glands are not punctured. Success is less likely on large or small fruits than on medium sized fruits. Stomatal infections are less frequent than infections at visible injuries. The organism multiplies in lesions on fruit of all sizes but does not increase but rather tends to disappear quickly in orange and lemon fruits inoculated after being picked at maturity. The organism seems to persist longer in mature grapefruit. Tests of cankers induced on young citrus fruits of orange, lemon, lime, and grapefruit were all negative when tested six or seven months later, after the fruit had matured. However, viable canker bacteria have been found in some of the tests of lesions on citrus fruits intercepted at U. S. ports.

In inoculated fruit viable bacteria may be present without forming a visible canker. Penicillium rot has a deleterious effect on the canker organism.

(10) Hasse, Clara H.
1915 Pseudomonas citri, the cause of Citrus canker. Journ. Agr. Res. 4: 97-100, pls. IX and X. Apr. 15, 1915.

Description of Pseudomonas citri Hasse and of its isolation in pure culture, inoculations, structure of cankers.

(11) Hill, Gerald F.
1918 History of Citrus canker in Australia with a brief account of the occurrence of the disease in the United States of America and South Africa. North. Terr. Australia Bul. No. 18, 8 pp., 8 pl. Oct. 1918.

The part dealing with South Africa is based on Miss Doidge's account in Sci. Bul. No. 8. A number of U. S. authors are cited. Hill became interested in the disease in Australia in 1912. After the cause of the disease was learned in 1916, various quarantine and eradication measures were undertaken. Details are given of the finding of citrus canker in individual holdings, severity of the disease on different hosts, the quarantine notice is given, and symptoms of the disease as it occurs in Northern Australia are given.

(12) Hino, I.
1931 The behaviour of the Citrus canker organism in the soil and in water. Studia Citrologica, Tanaka Citrus Exper. Stat. 4: 167-178. 1931 (English summary).

Canker organism remained viable several months in inoculated garden soil and in pond water so it could overwinter in the soil. The organism was found to thrive in relatively dry highly alkaline soils. Soil protozoa, which destroy bacteria including the canker organism, are not so active during winter.

(13) Lee, H. A.
1918 Further data on the susceptibility of Rutaceous plants to citrus canker. Journ. Agr. Res. 15: 661-666. Dec. 23, 1918.

This paper gives a tabulated summary of results of inoculations on plants of Rutacea representing 24 species in 20 genera, 19 species being more or less susceptible. *Fortunella hindsii* is suggested as possible original wild host.

(14) Lee, H. A.
1920 Behavior of the citrus-canker organism in the soil. Journ. Agr. Res. 19: 189-206. June 1, 1920.

The canker organism was found to disappear very quickly in normal soil under orchard conditions. When inoculated in sterilized soil the organism increases and multiplies. "The indication is that the normal soil organisms are antagonistic in some way to the existence of *P. citri* in the soil."

(15) Lee, H. A.
1922 Relation of the age of Citrus tissues to the susceptibility to Citrus canker. Philipp. Journ. of Science 20: 331-339, 4 pl., 1922.

Orange fruits of different types were tested and it was found that young fruits were quite susceptible while older fruits were slightly affected or immune. The period of susceptibility for grapefruit was longer than for oranges. Washington navels were virtually immune from stomatal infection after 85 days and wound infection after 110-120 days in one series of observations. Leaves also lost susceptibility with age.

(16) Loucks, K. W.
1930 Some physiological studies of *Phytomonas citri*. Journ. Agric. Res. 41: 247-258. 1930.

Citrus canker organism died out after 13 days in unsterilized Florida muck soils and after 6 days in unsterilized sandy soils but persisted for 150 days or longer in both types sterilized, inoculated, and kept at room temperature.

(17) Peltier, George L. and Frederich, William J.
1924 Further studies on the relative susceptibility to Citrus canker of different species and hybrids of the genus Citrus, including the wild relatives. Journ. Agr. Res. 28: 227-239 Apr. 19, 1924.

All grapefruit plants and all of the sweet orange group are susceptible to canker. All of the *Citrus nobilis* group show resistance. A considerable number of genera, species, and hybrids is listed and inoculation results noted.

(18) Peltier, G. L. and Frederich, W. J.
1926 Effects of weather on the world distribution and prevalence of Citrus canker and Citrus scab. Journ. Agric. Res. 32: 147-164, 1 map, 8 graphs. 1926.

Canker could develop in any citrus growing region but should be most severe in areas with the longest periods of temperature 80° F. or above. The precipitation curve is important, a fairly long period of plentiful moisture producing tender susceptible growth and humidity conditions favorable for infection.

(19) Stevens, H. E.
1915 Citrus Canker - III. Fla. Agr. Exp. Sta. bul. No. 128, pp. 3-20. Nov. 1915.

Historical summary, symptoms, studies confirming *Pseudomonas citri* Hasse as the pathogen and discussion of tests showing that *P. citri* lives for months in inoculated soil. Temperatures of 60° C. or higher for 5 minutes killed the citrus canker organism in all cases.

(20) Swingle, W. T., Robinson, T. R., and May, E.
1924 Quarantine procedure to safeguard the introduction of citrus plants: a system of aseptic plant propagation. U. S. Dept. Agr., Dept. Circ. No. 299, pp. 1 - 15, figs. 1 - 13. Jan. 1924.

This is a description and working plans of the special greenhouses and equipment developed to make it possible for the Department to obtain citrus breeding stock from foreign countries and be assured that no new diseases or insect pests were introduced with such stock and permitted to escape.

(21) Tanaka, T.
1918 *Citrus canker in Japan*: a translation of the first description of this disease, from the Japanese. "A new kind of

Pathogenic Microbe: being No. 8 of the organisms injurious to citrus." From "Nippon to Mikan," by Kumanosuke Abe, Pub. in M. 37, v. (May, 1904), p. 162-165. Phytopath. 8: 443-444, Aug. 1918.

Citrus canker was found to affect 70 to 80% of the 70,000 to 80,000 Washington navel orange trees in nurseries of the Meiji district of Japan in 1899. The disease attacked the new vigorous growth, spreading rapidly. Trees were not killed but the summer shoots were destroyed, causing a big loss to nurserymen. No infections were found on nearby Satsuma orange trees. The severity of the infection is more pronounced in the wet years and spreads more rapidly at such times.

Diseased parts should be removed and burned at once. Spraying with Bordeaux is recommended.

The organism was not isolated or described.

(22) Wolf, Frederick A.
1916 Citrus canker. Journ. Agr. Res. 6:69-99. 8 figs., pls. VIII-XI. April 10, 1916.

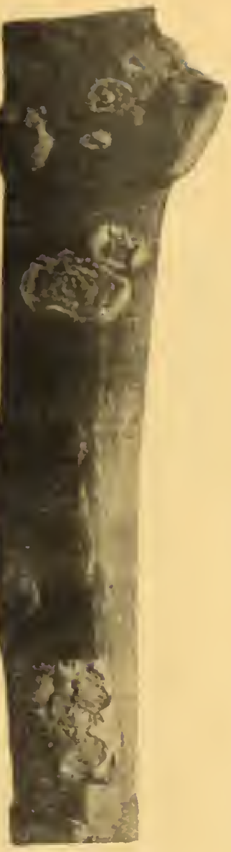
A detailed account of studies of the organism in culture and in the host, chemical changes in the host induced by the pathogen, dissemination, control. A fungus found to occur in the cankers caused by *Pseudomonas citri* is named *Phoma socia* and described. Complete eradication found to be the only effective control measure.

(23) Wolf, F. A.
1916 Citrus Canker. Ala. Exp. Sta. bul. No. 190, pp. 91-100. May, 1916.

The disease is discussed and illustrated, *Pseudomonas citri* Hasse illustrated and cross-sections of diseased tissue pictured. His tests indicated 65° C. temperature was required to kill the organism under some conditions. New infections may appear at any time during the growing season. Infections occur on old leaves and twigs as well as new. Old diseased areas may enlarge. Leaves are infected through stomata, which occur on the lower surfaces of the leaves, but only when a film of moisture is present. Infection also takes place through wounds. - Eradication is only known method of checking citrus canker.

Bacterium citri

Plate 1



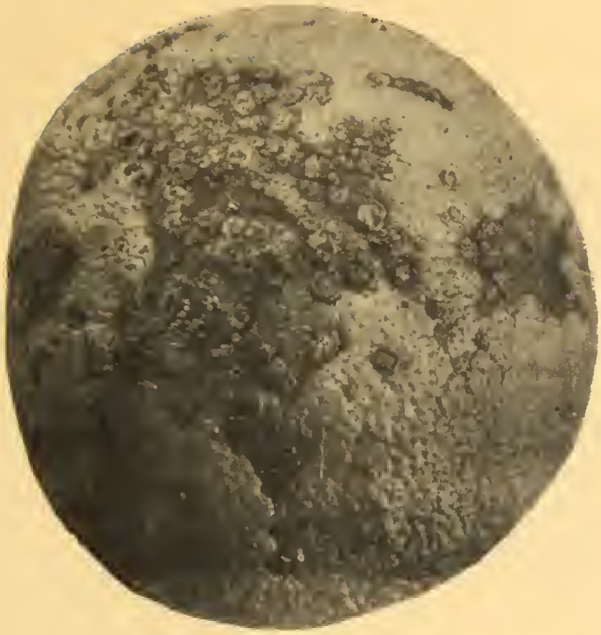


Fig. 1--
Infected grapefruit.

Fig. 2--Infected twig of *Citrus trifoliata*, showing broken membrane around lesions. (Magnified approximately x3).

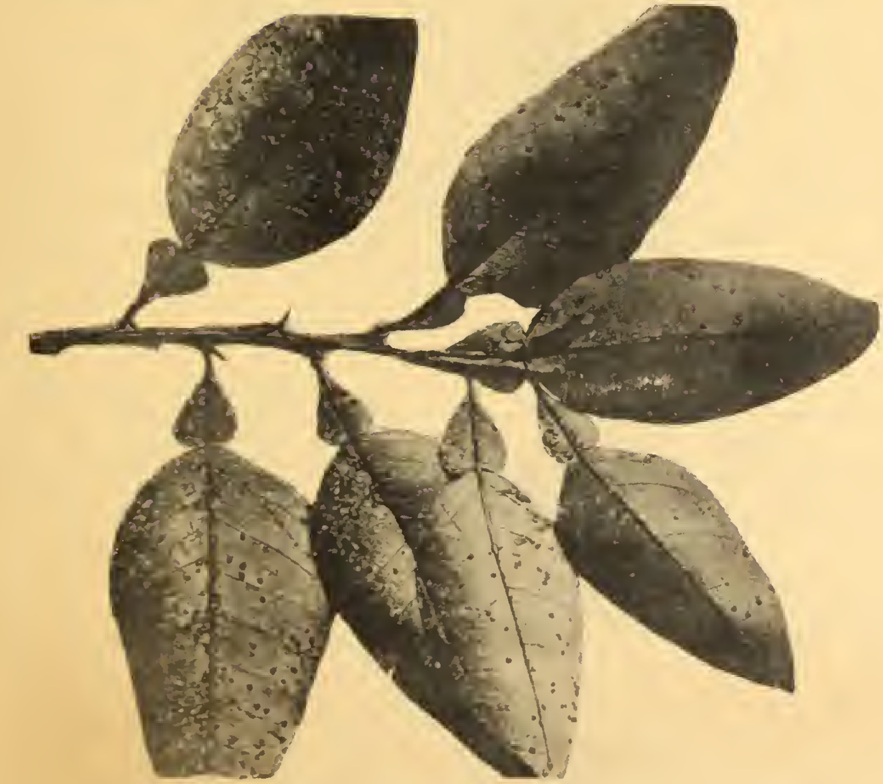


Fig. 3--Canker on grapefruit leaves.

(From Florida Bulletin 128. Fig. 4, 5, and 3.)

Bacterium citri

Plate 2

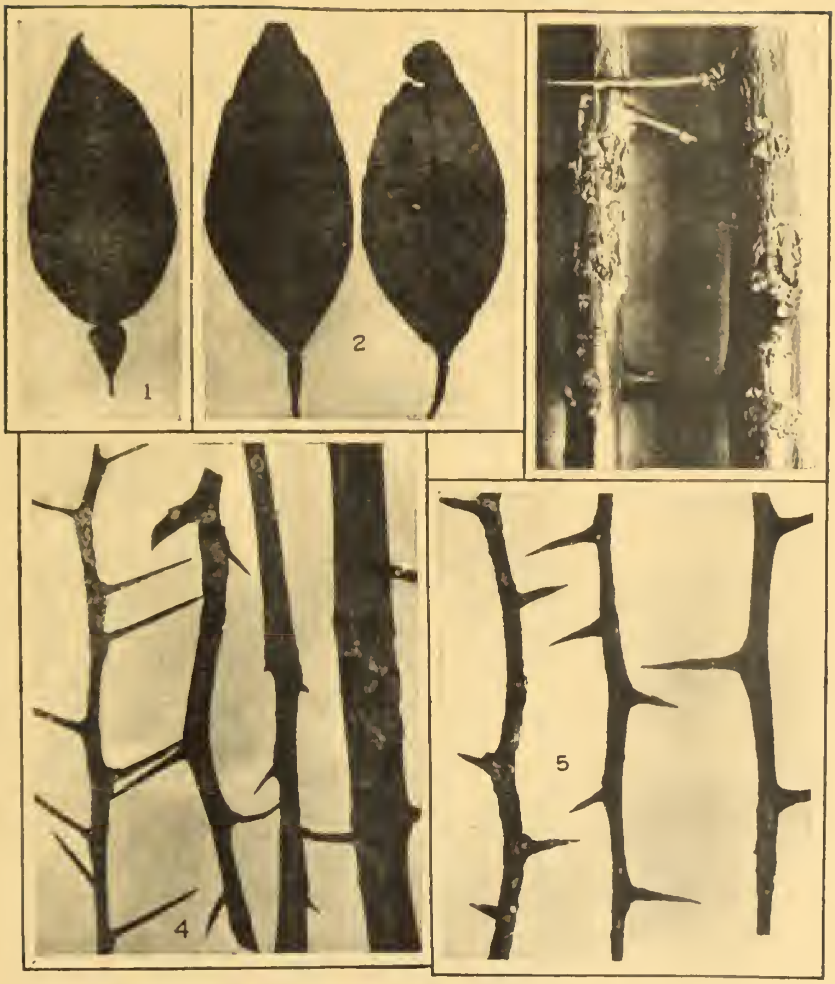


Fig. 1--Grapefruit leaf showing young Citrus cankers.
Fig. 2--Old Citrus canker on Satsuma leaves.
Fig. 3, 4. --Seedling grapefruit branches affected with Citrus canker.
Fig. 5--Severe canker infection of branches of *Citrus trifoliata*.

(From Jour. Agr. Res. 6: 69-100. Apr. 10, 1916. Plate VIII)

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